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OF

OPHTHALMOLOGY

APRIL, 1922

COMMUNICATIONS

ON THE MORPHOLOGY OF CERTAIN DEVELOPMENTAL STRUCTURES ASSOCIATED WITH THE UPPER END OF THE CHOROIDAL FISSURE*

BY

IDA C. MANN, M.B., B.S.

LONDON

PART I.

On the morphological value of certain cells found on the optic disc in human embryos.

ON examining sections through the centre of the developing optic disc in human embryos of about the eleventh week the appearances shown in Fig. 1 will be seen. It will be noticed that there is already a fair amount of differentiation of the retinal layers, and that nerve fibres are present. The nerve fibre layer, as it nears the disc, increases in thickness at the expense of the other retinal layers, which become thinner and blend with one another at the edges of the nerve. The nerve fibres, as they pass out of the eye, leave in the centre of the disc a small rather funnel-shaped interval, which is filled in with a cellular tissue, so that there is no real depression below the general surface of the retina here. Indeed, in some human embryos examined this small cone of cells formed even a slight elevation in the centre of the disc. In the figure a

*Read in the Section of Ophthalmology of the Royal Society of Medicine, February 10, 1922.

small blood vessel (a derivative of the intraneural portion of the arteria hyaloidea) can be seen at the apex of the cone. There are

FIG. 1.—Section through the optic disc of a 35mm. human embryo.

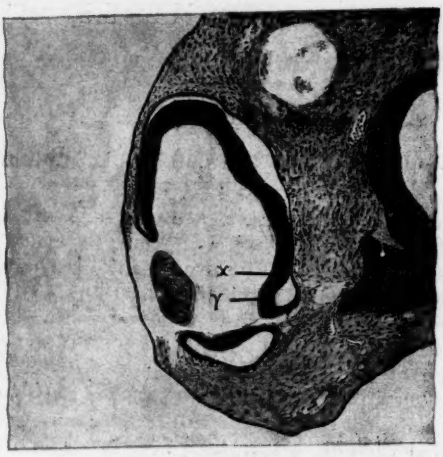

FIG. 2.—Section through the whole eye of a young chick embryo.

as yet no retinal blood vessels visible. Branches of the arteria hyaloidea can be seen faintly in the vitreous. It is to the consideration of the origin and morphological value of this small

cone of cells in the centre of the disc that the present paper is devoted.

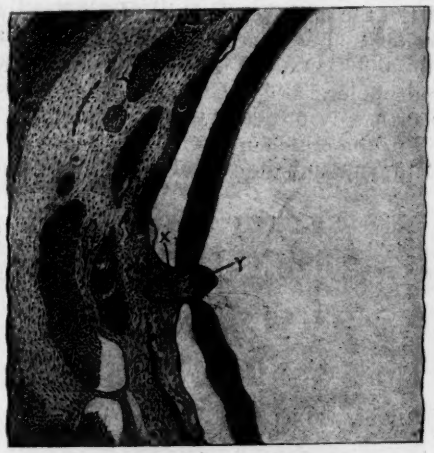

FIG. 3.—Section through the fused portion of the choroidal fissure in a chick embryo.

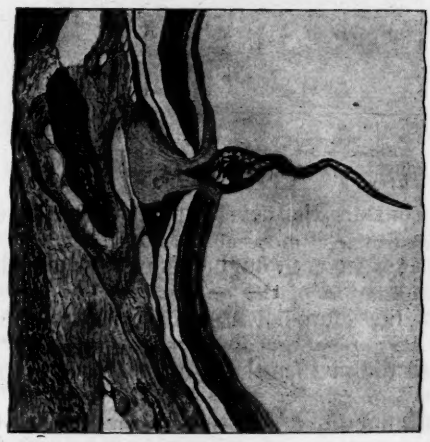

FIG. 4.—Section through the primitive pecten in a chick embryo

There are naturally two possible origins for these cells. First, they may, as has often been upheld, be mesodermal elements carried in along the choroidal fissure with the arteria hyaloidea.

They do not, however, resemble vaso-formative tissue, nor are they continued at all along the vessel in its passage through the vitreous. Secondly, they may be ectodermal in origin and derived from some part of the optic cup. It is, of course, not to be denied that a few mesodermal cells accompany the arteria hyaloidea and take part in the differentiation of its walls, but I think it can be shown that, morphologically at least, the cone of cells in the centre of the disc is primarily ectodermal in origin and is derived from the inner layer of the optic cup.

In order to understand the meaning of this cone of cells it is

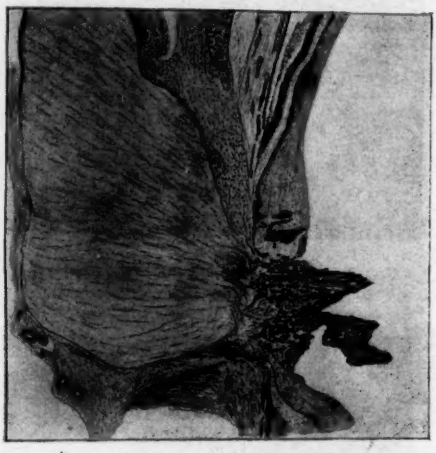

FIG. 5.—Section through the upper end of the pecten in a hen's eye.

necessary to consider the development of certain other animals, in which it is seen in a more elaborated form.

In looking at sections through certain early vertebrate embryos, in which invagination of the optic cup has occurred, but before the closure of the choroidal fissure or the appearance of nerve fibres, it will be noticed that the inner (retinal) layer of the cup is separated from the pigment layer and is thrown up into folds, as though it were of greater area than the pigment layer. This appearance of more rapid growth of the inner than the outer layer can be seen to a greater or less extent in most vertebrates, though it is absent altogether in certain primitive forms (e.g., the Ganoid, *Amia*) in which the inner and outer layers of the optic cup are everywhere in perfect apposition. From one point of view, then, developing vertebrate eyes can be divided into two groups, namely, those in which in early stages the inner layer of the optic cup

grows more rapidly than the outer, and those in which it does not. It is with the first only of these two groups that we are concerned. In this group the overgrowth of the inner layer leads to a heaping up and eversion of this unpigmented layer along the choroidal fissure. This is most marked in the upper part of the fissure, since the overgrowth is apparently more active at the posterior pole of the eye than further forward. If the overgrowth is only moderate there will be eversion of the inner layer along the upper part of the choroidal fissure, so that, when fusion takes place between the lips of the fissure here, the actual areas which fuse are derived

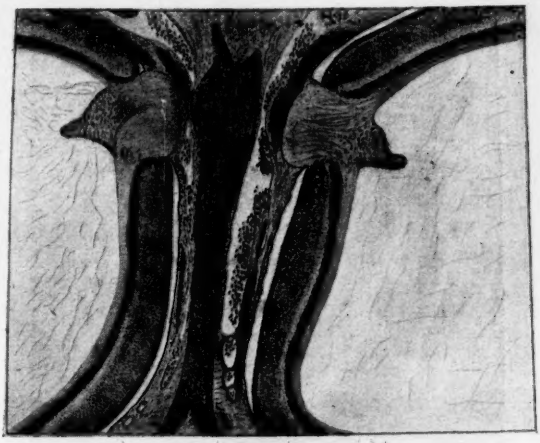

FIG. 6.—Section passing through portions of both eyes in a Calotes embryo.

from inner layer only and are free from pigment. There will therefore be after closure a linear non-pigmented " scar " on the outside of the cup in the line of the fissure. If, on the other hand, the overgrowth of the inner layer is greater than this, this layer will be thrown up into more marked folds along the edges of the fissure, and when fusion occurs two ridges will be seen in the line of the fissure, both derived from the heaped-up inner layer, one on the outside of the optic cup showing as a well-marked non-pigmented ridge interpolated in the otherwise uniform pigment epithelium and running down from the point of attachment of the optic stalk in the line of the closed fissure; the other a ridge in the same line, but projecting into the interior of the cup. This heaping up of the inner layer in the line of the fissure inside the eye I have called the *crista intraocularis*. This structure is very well marked in birds and its development and fate in the chick will now be considered.

Fig. 2 shows a section through the eye of a young chick embryo. The section passes through the choroidal fissure just below the insertion of the optic stalk. It will be seen that the pigment layer is only one cell thick and that the inner (retinal) layer is already several cells thick, and is separated from the pigment layer. At the fissure the heaping up and eversion of the inner layer is very apparent, especially at the upper edge, the inner layer becoming continuous with pigment layer at X, some distance from the fissure and outside the eye. The heaping up which, after fusion, will form the internal ridge (*crista intraocularis*), is seen at Y. A filmy

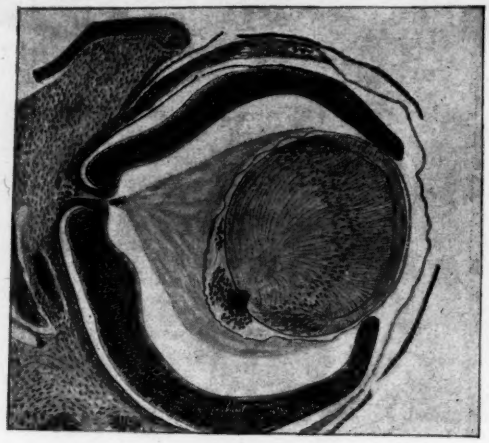

FIG. 7.—Section through the whole eye of an Acanthias embryo.

strand of mesoderm containing blood corpuscles passes in at the fissure and stretches across the lower part of the cup at this stage. Fig. 3 shows a section through the upper (just closed) part of the fissure of an older chick. It will be seen that the two ridges derived from the heaped up inner layer are well marked. On the outside of the eye the everted portions of the inner layer have fused, forming a non-pigmented projection continuous with the pigment layer at X, which corresponds with the point X in Fig. 2. The *crista intraocularis* projects as a definite papilla into the vitreous. The continuity between and similarity of the cells of the inner layer of the cup and the cells of the *crista intraocularis* can be seen at this stage, when very little differentiation has occurred in either. The cells of the external everted projection can also be seen to resemble those of the inner layer and show a definite epithelial arrangement,

which entirely differentiates them from the surrounding mesoderm outside the cup. There are no blood vessels or corpuscles visible in either of the two projections at this stage, and I think it is quite obvious that both of them are in continuity with and derived from the walls of the optic cup and are not mesodermal in nature. In this section nerve fibres are just beginning to appear on the surface of the retina, and are running towards the site of the fissure, along the line of which in its upper part they leave the eye. In doing this they do not grow over the vitreous surface of the *crista intraocularis*, but pass through deeply at its base, running obliquely

FIG. 8.—Section through the optic disc in a young dogfish.

and thus cutting off the central cells of the *crista intraocularis* from the rest of the inner layer, though the original continuity can long be traced through scattered nuclei lying among the nerve fibres and connecting the base of the *crista intraocularis* with the end of the inner layer. Thus the *crista intraocularis* becomes apparent as a wedge of cells projecting into the vitreous between the converging bundles of nerve fibres, and at this stage corresponds with the cone of cells seen in the human embryo in Fig. 1, although in the chick the crista is linear and not circular. Bergmeister has described this wedge of cells in the chick but does not, as far as I am aware, trace their subsequent fate. Ucke, following up Bergmeister's work, again maintains their ectodermal origin and further shows the existence of similar ectodermal cells on the disc in certain fish and amphibians and in the sheep. He also mentions

that in certain low forms (*Ammocoetes*) the nerve fibre layer is not the most internal in the definitive retina, but is covered by a double layer of epithelial cells, and he suggests that the cone of cells seen in the chick's eye represents all that is left in higher forms of this internal epithelial layer. However that may be in *phylogeny*, the fact remains that *ontogenetically* the *crista intraocularis* is composed of cells cut off from the inner layer of the optic cup by issuing nerve fibres.

As the cleft closes (in the chick) the small strand of vascular mesoderm which was seen entering the eye through the fissure in

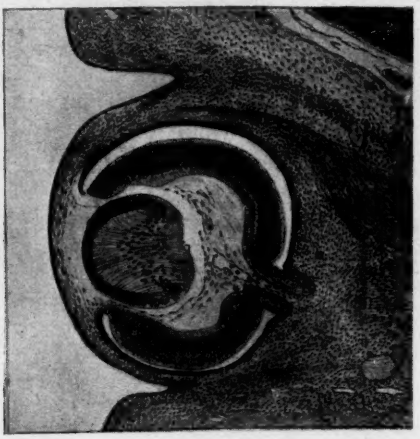

FIG. 9.—Section through the whole eye of a pig embryo.

Fig. 2 gets pushed more and more distally. It eventually disappears, and birds do not possess a definite arteria hyaloidea. From its proximal end in the eye, however, branches pass into the lower end of the *crista intraocularis* and vascularize it from below upwards. The *crista intraocularis* then grows out into the vitreous as a thin wedge of very vascular tissue, and forms the primitive pecten. This continues to grow and becomes many times folded upon itself. Later, pigment is developed in it, but long after pigmentation of the other structures in the eye is complete. Fig. 4 shows a section through the eye of an older chick. It will be seen that the *crista intraocularis* is now entirely cut off from the rest of the inner layer and that it is extremely vascular and is beginning to show signs of folding. Fig. 5 shows a section through the extreme upper end of the pecten in an adult hen's eye. It will be

seen that its position corresponds with that of the *crista intraocularis* in the embryonic chick's eye. The pecten is thus primarily an ectodermal structure derived from the inner layer of the optic cup, and is secondarily vascularized by (mesodermal) vessels growing into it from its lower end, these being derived from the proximal end of the strand of mesoderm which enters the open part of the choroidal fissure at an early stage and disappears without forming a definite hyaloid artery.

The vascular supply of the pecten is separate from that of the choroid, as can be shown both by a study of its development and

FIG. 10.—Section through the whole eye of a 15mm. human embryo.

by experiments such as those of Mihalkovics, who found that, on injecting the choroidal vessels, no fluid passed into the pecten. He denied the truth of the opinion of Gegenbaur and Leydig who looked on the pecten as an intraocular portion of the choroid, but he considers it as developed from mesoderm caught between the lips of the closing fissure. This view is also upheld by Slonaker, who has studied the development of the sparrow. It is, I think, certain that the pecten is primarily an ectodermal structure, since a definitive *crista intraocularis*, continuous with the inner layer of the optic cup, can be seen before there is any sign of vascularisation. In the adult condition, it is true, the mesodermal elements dominate the picture; the ectodermal remnants only remain as a neuroglial network supporting the capillaries, and continuous with the neuroglial septa in the optic nerve. This view is held by

Denissenko, who, however, gives no definite account of the development, and by Franz, who arrived at the conclusion of the nervous origin of the pecten from a study of the adult organ only.

Fig. 6 represents the developing pecten in the lizard *Calotes*. The optic discs of both eyes are shown. The condition is exactly similar to that seen in the chick (Fig. 3) and again there is as yet no vascularization of the *crista intraocularis*, which shows here a very definite epithelial arrangement of its surface cells. In *Calotes*, as in the chick, a vessel enters the vitreous through the open part of the fissure below the *crista intraocularis*.

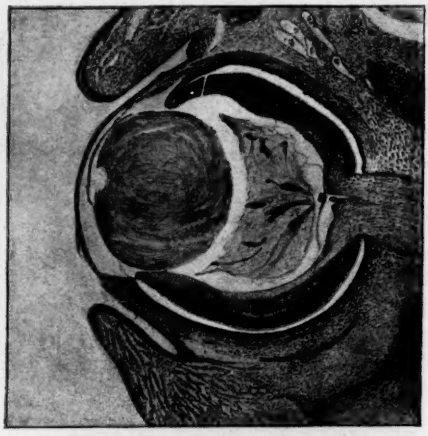

FIG. 11.—Section through the whole eye of a 33mm. human embryo.

The final elaboration of the *crista intraocularis* is therefore the pecten of lizards and birds. We will now turn to other forms in which a definitive pecten is never developed. Among fishes the dogfish, *Acanthias*, will serve as an example. Fig. 7 shows a section through the open choroidal fissure in an *Acanthias* embryo. It will be seen that the overgrowth of the inner layer is not so marked as in the chick, but that there is definite separation between the layers and heaping up of the inner layer in the lips of the fissure. Fig. 8 shows a section through the optic disc of a much older dogfish, in which some cells of the inner layer have been cut off by the issuing nerve fibres, and remain in the centre of the disc as a cone-shaped plug. Nuclei can still be seen scattered among the nerve fibres and connecting this plug with the inner layer, of which it is part. Since the nerve fibres only leave the eye at the upper end of the choroidal fissure in the dogfish, this plug of cells (the analogue of the *crista intraocularis*) is circular

and not linear, and since the vascular mesoderm entering the fissure in Fig. 7 entirely disappears and does not send branches to these cells, the cone does not grow out into the vitreous or undergo any further development at all.

Among mammals the same condition of overgrowth of the inner layer of the optic cup is found. In some (e.g., mouse) it is only great enough to produce eversion and the appearance of non-pigmented cells on the outside of the cup, while in others (pig and man) an elevation (*crista intraocularis*) is formed inside the eye as well. Fig. 9 shows a section through the eye of a young pig

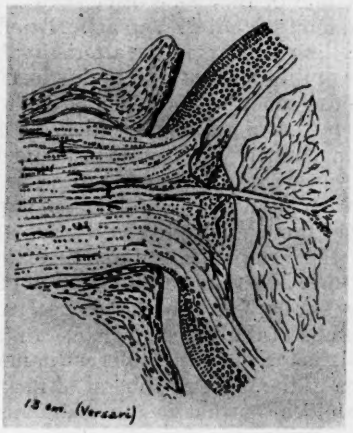

FIG. 12.—Diagram after Versari to show the development of the arteria centralis retinae.

embryo just below the optic stalk. The fissure is open, and it will be seen that the inner layer is markedly everted and also slightly heaped up inside the eye. When fusion occurs, and nerve fibres appear, a few of the cells of the inner layer are cut off and remain as a cone-shaped plug in the centre of the disc. In man a similar condition is seen. Fig. 10 shows the eye of a human embryo of the sixth week. It will be seen that eversion of the inner layer is well marked in the fissure; the internal heaping up is not so well marked. The hyaloid artery is seen entering the fissure. When fusion occurs and nerve fibres appear a few of the inner layer cells are cut off just as in other animals and remain as a cone-shaped plug on the disc. In addition, however, in man (and pig also) the vessel entering the eye remains as the very definite arteria hyaloidea. Fig. 11 shows the condition in a nine weeks human

embryo. The optic nerve is forming and a definite nerve fibre layer is apparent. In the centre of the disc the arteria hyaloidea is seen entering the vitreous at the bottom of a little depression. On either side of this depression are a few very definite cells, the representatives of the *crista intraocularis*, namely, cells cut off from the inner layer of the optic cup by the issuing nerve fibres. In this section the connection of these cells with the retina rather than with the vessel is very clearly shown, since they are separated from the vessel by a definite space, but are continuous with the nerve fibre layer and with scattered cells in this which appear to be derived from the other layers of the retina.

The subsequent fate of this cone of cells in man is of interest. It has been worked out in detail by Versari. Briefly it is as follows. A bulb-shaped swelling appears in the arteria hyaloidea as it passes through the centre of this cone, and from this bulb branches appear which vascularize the cone. This is analogous to the vascularization of the *crista intraocularis* in the chick by derivatives of the vascular mesoderm entering the vitreous at its lower end. The cone does not, however, grow outwards into the vitreous in man, but the vessels in it insinuate themselves among the nerve fibres and, spreading over the whole retina in the substance of the nerve fibre layer, form the arteria centralis retinae and its branches. Fig. 12 from Versari shows the vascularization of the cone. This occurs in the pig similarly. In the mouse, where there is no cutting off of the cells of the inner layer to form a cone, the intraocular blood supply is formed quite differently, namely, by a laying down on the retina of the most peripheral of the vitreous branches of the arteria hyaloidea, so that in these animals the branches of the arteria centralis retinae are always superficial to the nerve fibres and not embedded in them as in man.

It is thus evident that the cells found on the optic disc in human embryos are ectodermal cells of the inner layer of the optic cup, which have become cut off from their fellows by issuing nerve fibres. They are the representatives of the cells of the crista intraocularis in birds, and their function both in man and in birds is to form a nidus for the proximal branches of the vessel from which the definitive intraocular blood supply is developed. In birds the cells of the crista are more definitely cut off from the rest of the inner layer, and so, when it becomes vascularized, it grows out into the vitreous, while in man and some other mammals (e.g., pig) the cells are more scattered among nerve fibres, so that when vascularization takes place the small vessels grow in amongst the nerve fibres and penetrate in this layer all over the retina. In animals in which there is no intra-retinal blood supply (e.g., *Acanthias*) the cone of cells may still be present but the arteria hyaloidea disappears without vascularizing it.

PART II.

On the morphological value of certain non-pigmented cells found below the insertion of the optic stalk in the 15mm. human embryo.

FIG. 1 represents a section through the optic cup of a 15mm. (sixth week) human embryo just below the insertion of the optic stalk. The inner layer of the cup is everywhere separate from the outer layer, and the cells of the outer layer show dense pigment deposited in them, except at the posterior pole, where it is to be seen that

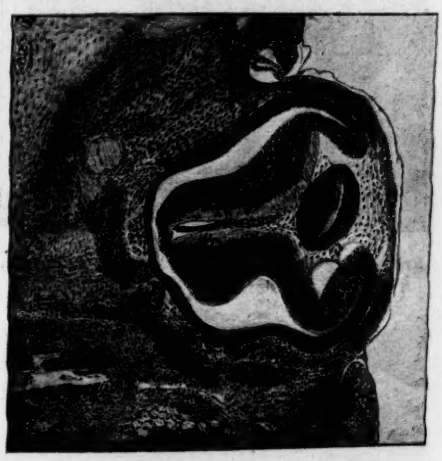

FIG. 1.—Section through the whole eye of a 15 mm. human embryo.

a small mass of unpigmented cells interrupts the pigment layer. This mass marks the situation of the upper end of the closed part of the choroidal fissure, the only open part of this upper end being at its extremity for the passage of the hyaloid artery. The development of this unpigmented mass of cells is straightforward enough and it is easy to see the ontogenetic reason for their presence. Their morphological associations are, however, interesting.

As I pointed out in the first part of this communication, there is in certain vertebrate eyes a more rapid growth of the inner layer of the optic cup (at the posterior pole) than of the outer. If this occurs before closure of the choroidal fissure, it leads to eversion of the inner layer in the lips of the fissure, most marked in its upper part, so that, when closure of the fissure occurs, fusion takes place between unpigmented (inner) layers only. Thus it comes about

that there is an unpigmented "scar" (everted inner layer) visible on the outside of the pigmented optic cup for some little time after

FIG. 2.—Section through the eye of a 15 mm. human embryo just above that shown in Fig. 1.

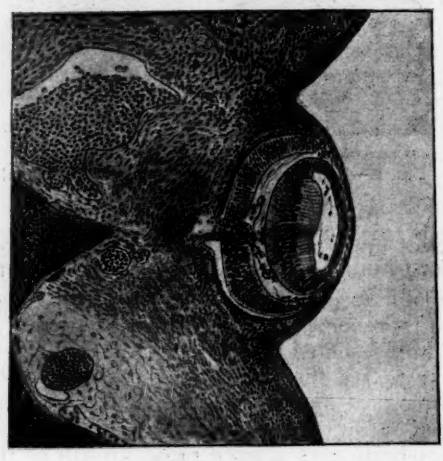

FIG. 3A.—Section through the whole eye of a mouse embroyo.

closure of the fissure. This is the case in man in the upper portion of the fissure. Fig. 2 shows a section through the same eye as Fig. 1, very slightly higher up, namely through the still open

upper end of the choroidal fissure. It will be seen that the inner layer of the cup, which is still undifferentiated, is separated from

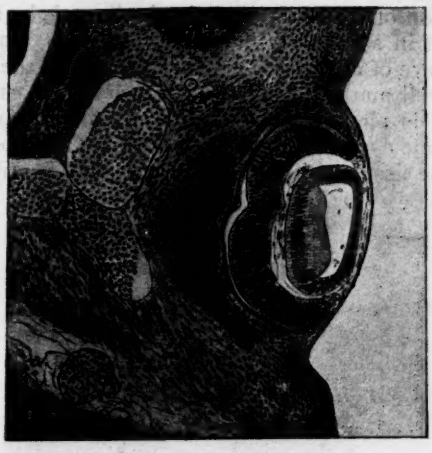

FIG. 3B.—Section through the whole eye of a mouse embryo.

FIG. 4A.—Section through the whole eye of a pig embryo.

the pigment layer and is thrown up into folds and is also pushed out considerably in the lips of the fissure. It is obvious that fusion will occur between these everted non-pigmented areas and

that, when subsequent separation into outer and inner layers takes place in the region of this fusion, a portion of the inner layer will be left in continuity with the pigment layer.

The eversion of the inner layer in the lips of the fissure is even more marked in some other mammals. Fig. 3A shows a section through the eye of a mouse, in the upper part of the fissure. The eversion is well marked. There are no nerve fibres in the retina, but pigment is already developed in the outer layer. Fig. 3B shows the same eye at a lower level, through the closed peripheral portion of the fissure. The line of the fissure is still evident as a

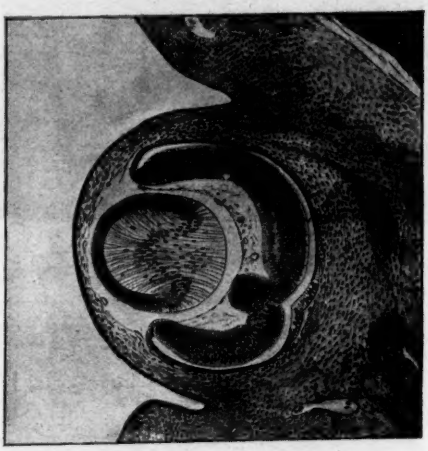

FIG. 4B.—Section through the whole eye of a pig embryo.

slight groove on the outside and ridge on the inside of the cup, and the unpigmented everted portion of the inner layer is very obvious. It is in direct continuity with the pigment layer and has lost all connection with the rest of the inner layer.

Figs. 4A and B show exactly similar sections through an embryonic pig's eye. The eversion is even more marked, and, in this case also, the connection of the unpigmented cells with the inner layer is rapidly lost.

A similar condition is found in the Sauropsidans. Fig. 5A shows a section through the whole eye of a *Chrysemys* embryo (terrapin). It will be seen to resemble closely the eye of the young chick figured in Part I (Fig. 2, Pt. I). There is marked eversion in the fissure. Fig. 5B shows an older Chrysemys after fusion has occurred along the whole length of the fissure. Its site, however,

is clearly marked by the small mass of unpigmented cells in the outer layer in the lower part of the eye.

FIG. 5A.—Section through the eye of a Chrysemys embryo.

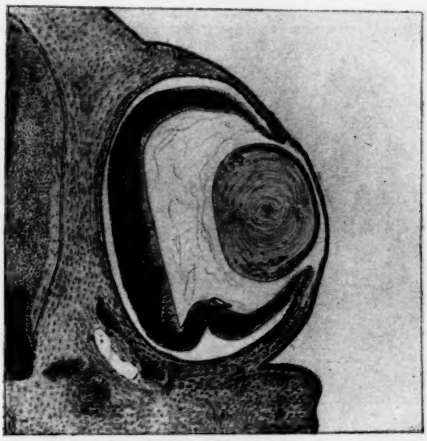

FIG. 5B.—Section through the eye of a Chrysemys embryo.

In the chick the condition is further specialised and elaborated. As was pointed out in Part I the overgrowth of the inner layer produces not only eversion along the fissure, but also the heaping

up of a "*crista intraocularis*" inside the eye. After closure of the fissure, therefore, two ridges are apparent in its line, one pro-

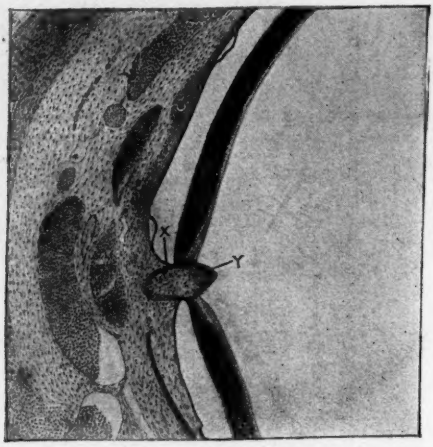

FIG. 6.—Section through the eye of a young chick embryo.

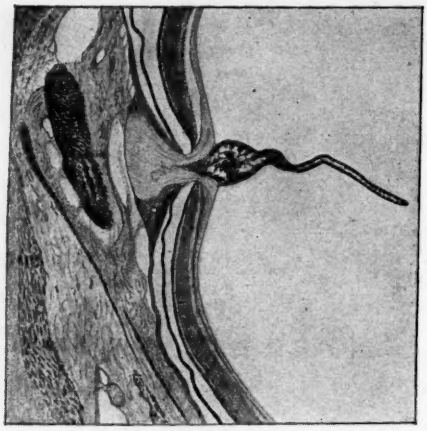

FIG. 7.—Section through the primitive pecten in a chick embryo.

jecting into the eye (*crista intraocularis*) and the other unpigmented, on the outside. In this bird the outer ridge does not lose its connection with the rest of the inner layer as it does in the other

animals considered, with the result that when nerve fibres develop in the retina (which they do in the chick very soon after fusion has begun) they pass into this everted projection as they leave the eye, since it is in origin directly continuous with the inner (fibre-bearing) layer of the cup. The nerve fibres, then, leave the eye in the line of the upper part of the closed fissure. They enter the everted projection and run up in the substance of this on the back of the eye in order to pass into the trunk of the nerve at the level of the upper end of the choroidal fissure. These bundles of fibres running up on the back of the eye constitute the " cauda " of the adult hen's nerve. Fig. 6 shows the site of the fissure in a chick's eye just when nerve fibres are appearing. They are seen to be passing into the everted non-pigmented portion, cutting off, as they do so, the inner ridge (*crista intraocularis*) which will later form the pecten. Fig. 7 shows a later stage, when the cauda appears as a bundle of nerve fibres, the original inner layer cells having disappeared, and the pecten is growing out into the vitreous.

Thus we see that the little mass of unpigmented cells seen on the outside of the human embryo's eye at about six weeks, is the remains of that portion of the inner layer which became everted in the lips of the fissure before closure. In the chick this occurs over a large area and nerve fibres growing into it form what is known as the " cauda " of the nerve, but in mammals (including man) the eversion only takes place at the upper end of the cleft, no nerve fibres grow into it, and the little mass of cells merely represents for a short time an abortive potential cauda. Under normal conditions it disappears rapidly and completely.

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KERATOMALACIA IN SOUTHERN INDIA

BY

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OF recent years a considerable amount of attention has been drawn to deficiency diseases. Various accessory food factors have been proved to be essential to the animal organism, and experimental deprivation of these substances has given rise to recognizable pathogenic states. Amongst these the experimental condition known as keratomalacia or xerophthalmia has been shown to be due to a deficiency of fat-soluble A, but so far as can be judged from the literature it is still questionable whether the clinical condition described under these names is similarly produced.

It may be well in the first instance to refer to the nature of experimental keratomalacia.

McCollum[1] and his co-workers proved that in the case of white rats, the specific result of a lack of fat-soluble A in the diet is xerophthalmia. It is usually developed about the fortieth to fiftieth day of deprivation. He states, " the eyes become swollen so badly that they are opened with difficulty or not at all. The cornea becomes inflamed, and unless the missing dietary essential is supplied blindness speedily results." He obtained complete recoveries within two weeks of starting butter fat 5 per cent. in the diet, provided the animals were not previously blind. Normal eyelids could be restored even if the cornea was destroyed.

Professor Hopkins[2] describes keratomalacia or xerophthalmia as consisting primarily and essentially in an abnormality of the corneal tissue which exhibits an exceptionally low resistance to a specific deficiency. He says, " the onset is sudden and is first noticed as an opacity but the symptoms develop so rapidly that it is hard to follow their sequence," within a few days there is complete panophthalmitis. He draws attention to the facility with which the experimental condition may be stopped with a little cod liver oil.

Secondly, in order to view the subject in its relation to human food, it is necessary to consider the sources of fat-soluble A, the possible effects of diets deficient in this substance, the influence of other substances such as salts and protein in the diet, and the likelihood of destroying fat-soluble A in the preparation of articles for food.

One gathers from the perusal of McCollum's work that there is very little fat-soluble A in diets other than those which contain green leafy vegetables and fat of milk, and that the great sources of this dietary essential are milk, butter, eggs, green leafy vege-

tables and glandular organs. Diets composed of seeds and their products, tubers, roots, meats, etc., in the amounts in which they are ordinarily consumed do not furnish enough of this substance for the maintenance of an optimum state of well being. They may, however, when other deficiencies are corrected, contain enough fat-soluble A to induce fairly good growth and prevent xerophthalmia. Diets made up largely of substances poor in fat-soluble A do not supply enough of this substance to prevent xerophthalmia indefinitely, or under the stress of lowering conditions such as pregnancy. Such diets bring an animal into what McCollum calls the " twilight zone," where small shifts in the quality of the diet with respect to any factor may determine the pathogenic state. Another element to which he calls attention is the digestive disturbance caused by bad fare, which may beget xerophthalmia not due to deficiency in the diet, but to the impaired vitality. He makes the interesting observation that fat-soluble A may be reduced to a certain minimum without producing xerophthalmia, whereas the same intake of this substance will not prevent the characteristic eye trouble when the intake of protein is likewise sufficiently lowered. A vegetarian diet without supplementary calcium, sodium, chlorine, will give rise to stunted growth, but a diet of cereals satisfactorily supplemented by salts of these elements and a protein of good quality, may for a long period prevent the onset of disease. The influence of salts in general, and those of calcium and sodium in particular is of great importance in association with border line dietaries, although their direct influence in keratomalacia is not indicated. In this connection Sir James Barr[2] directed attention to the inorganic constituents of the blood, and the rôle of calcium and other salts in maintaining physiological equilibrium. He appeared to think that in the modern hue and cry after vitamines, the great importance of the inorganic constituents of the diet was overshadowed, and he indicated that in the consideration of such a complicated process as that of human metabolism, the interdependency of all factors had to be reckoned with. With regard to the stability of fat-soluble A, drying and heat (as in ordinary cooking) do not appear to destroy it, but oxidisation easily alters its nutritional qualities.

Long before the experimental keratomalacia or xerophthalmia of animals was described, clinicians in many parts of the world had recorded pathogenic states of human beings under these names. It may well be asked, what is the relation between the two, if any? The opinions of some experimental and clinical observers on this point are worth noting.

Professor Gowland Hopkins[2] expresses the opinion that the above condition as met with in deficiency feeding may have no

clinical counterpart. He observed that the experiences of Bloch in Denmark that it has were not generally accepted by clinicians. He considered that protection was given by a very small supply of the special factor, and he assumed that if there is any possibility of a condition with a similar origin occurring in human beings, this fact might account for its rarity. McCollum[1] observes that it is important to enquire whether or not this disease has ever occurred in man, and refers to Mori's description of xerophthalmia in children in a time of food shortage. He considers that Mori's cases should be looked upon as a " deficiency disease " not hitherto recognised in its proper relation to diet. He states, " it is not a fat starvation, but if it be the same condition which McCollum and Simonds have definitely shown to be readily relieved in its early stages by the administration of such food stuffs as contain liberal amounts of fat-soluble A, it would not be relieved by feeding with vegetable fats in any amount." He leads one to infer that it ought to be relieved by feeding with foods rich in fat-soluble A.

Hess and Unger[3] are of opinion that the danger to infants of a diet deficient in fat-soluble A is slight, provided it includes sufficient calories and is otherwise complete. They considered that in spite of the fact that this vitamine is not widely distributed in nature, a disorder that may be termed " fat-soluble deficiency," marasmus, or xerophthalmia is hardly to be apprehended from a clinical standpoint. They point out the importance of other elements in the diet in controlling disease, and that fat-soluble A is not the controlling influence when considered alone. In their experimental work on children, health and vigour were maintained despite amounts of fat-soluble vitamine so small as rarely to be encountered in times of peace. Hess[4] failed to produce xerophthalmia in children deprived of fat-soluble A for periods of eight and nine months, nor did they develop rickets. He concluded from the experimental feeding of children that dangers from a lack of this dietary factor need not be apprehended if the diet is otherwise complete.

McCarrison[2] remarks on the frequency of the affection in Madras where he had seen a number of cases under the care of Lieut.-Colonel Kirkpatrick, once Superintendent of the Government Ophthalmic Hospital. Bloch[5] described necrosis of the cornea with ulceration in children in whom recoveries followed feeding on breast milk or whole milk and cod liver oil.

Czerny and Keller[6] describe a condition of the eyes in children suffering from malnutrition as a result of being restricted to a cereal diet.

Consideration of the various opinions recorded gives one the impression that there is a certain amount of confusion as to the

relation between the keratomalacia of man and that of animals, and also between the different conditions described under this title in man. The term is perhaps a little unfortunate as it tends to limit one's attention to the eye. A widely circulated standard description of the human condition known as keratomalacia is that given in Fuchs' Text-book of Ophthalmology, 6th edition. There are numerous other records which need not be referred to here, but in order to avoid any misconception as to what the term keratomalacia implies when used with reference to cases in the Ophthalmic Hospital, Madras, a somewhat lengthy presentation of the subject is given below.

To begin with, Colonel Elliot, in his recent text book[7], under the heading "Night-blindness," makes it quite clear that the corneal trouble is only one of the manifestations of a great general disorder of a nutritional type. In the title to the chapter he draws attention to another marked symptom of the same disorder. He not only deals with the condition as observed by himself, but gives a very extensive reference to literature on the subject, so as to make it possible for comparison to be drawn between the conditions of this type occurring in other countries.

Colonel Kirkpatrick has furnished me with the following note on the subject, embodying his ideas not only of the clinical features, but also of the treatment, which I give *in extenso*:—"This condition occurs with considerable frequency in the South of India, and it is rare for the children's ward of the Madras Government Ophthalmic Hospital to be without some examples of the condition. It is seen most often during the periods of scarcity and famine, when all diseases due to lack of nutrition are common. Children under the age of five are most commonly affected, but it may be seen in older children and even in adults, especially those who are suffering from chronic inflammatory diseases of the intestine. Affected children have usually been bottle-fed on unsuitable food, or breast-fed by mothers whose nutrition is in a low state. It is quite common for Indian children in the South to be suckled until three or four years old (unless the arrival of another infant cuts off the available supply), and this appears to affect them deleteriously and to predispose to the disease. Keratomalacia may also be brought about in young children by the exhaustion which follows any acute disease. The principal factor in the causation of the disease appears to be the lack of some essential substances in the dietary, or, if such are present, to an inability to assimilate them. Experience has shown that these substances (or the most important of them) are present in cod liver oil, as its beneficial effect, if absorbed, is undoubted, but their exact nature is uncertain as yet. The

characteristic feature of keratomalacia is a necrosis of the substantia propria of the cornea. This necrosis appears to be independent of a superficial microbic infection, and is not accompanied by any excessive vascular reaction. In many respects it would appear to be a coagulation necrosis of the tissue. A personal impression (which has no value as evidence), has been formed that the thyroid gland and the liver are affected adversely by a food deficiency and that the changes in the cornea are largely the result of alteration in the secretions of these glands.

A premonitary sign of keratomalacia is the occurrence of a condition which closely resembles, and is probably identical with, the ordinary night-blindness. Some stiffening of the triangular area of conjunctiva on each side of the cornea is found. The area corresponds to the part of the bulbar conjunctiva which is exposed by the palpebral aperture and the base of the triangle is formed by the limbus. The changes are best marked on the temporal side. The disease affects both eyes. Even at this early stage the hair often appears dry and the surface of the skin roughened. The triangular patches are at first darker than the surrounding conjunctiva, but soon show a white formation like sea-foam or soap-lather, on their surfaces. Some general darkening of the bulbar conjunctiva is generally present. As the disease progresses, the hair becomes more dry and scanty, and has a peculiarly bleached appearance; the skin also looks dry, rough and earthy. The pigmentation of the bulbar conjunctiva deepens and a faint grey haze appears between the centre and the periphery of the cornea, most commonly on the temporal side. No vascular reaction accompanies the appearance of this haze, which is situated in the substantia propria. The haze spreads and the whole cornea is soon involved. A well-marked icteric tinge is often now added to the pigmentation of the conjunctiva. The corneal surface next becomes dulled and the haze assumes a yellowish tint. A hypopyon may form at this stage. The tissues of the cornea now appear to melt away like thawing snow, and the entire structure yields *en masse* to the intraocular pressure and is cast off, so that the whole of the iris is exposed. The lens is next extruded and the vitreous lies bared in the pupil, giving the latter a jet-black appearance. A low type of inflammation is likely to set in now, or death, caused by the general condition, may take place. If the local condition is allowed to run its course, healing may sometimes occur and phthisis bulbi be the final result.

Cleanliness, warmth, and protection are the essentials in the local treatment of the disease. The conjunctival sac may be flushed out with warm normal saline solution twice or thrice daily and a drop of 10 per cent. argyrol or 0.75 per cent. nitrate of silver

solution instilled. The conjunctiva should be kept constantly lubricated by frequent instillations of castor oil. Dry heat is often useful, and this may be applied by a bran bag (dry bran heated on a metal plate over hot coals or charcoal and then placed in a flannel bag), or a Japanese hand warmer; these are held over the eye in such a way as to exert no pressure upon it. The hot air from an electric blower, if such an instrument is available, may be directed on the closed lids. Atropin is generally harmful, as it may excite conjunctival irritation, and it is unnecessary as iritis is nearly always absent. If it is considered advisable to dilate the pupil, hyoscin may be used with caution.

The constitutional treatment is the most important factor in staying the progress of the disease. If diarrhoea is present (as it generally is), a full dose of castor oil may be given; this is followed by the administration, every two or three hours, of an emulsion containing 10 to 20 minims of castor oil, according to the age of the patient, until the stools become yellow and the mucus disappears from them. Fair results were obtained by the use of treatment on these lines followed by a mixture which contained the chlorides of calcium, sodium, and ammonium. This mixture was prescribed in the earlier cases owing to an impression that calcium and the chlorides were deficient in the food, and it still appears to be of benefit. Cod liver oil is, however, the essential remedy for the disease; it should be given with considerable care as the patient may not be able to digest it, and in such a case it should be rubbed into the skin of the axillae, back, and abdomen, but it should be administered by the mouth when it is at all possible to do so. There is often a scorbutic taint in this disease, and fruit juice should be given very freely. Whether caused by the intestinal disease or by the food deficiency, the thyroid gland is often atrophic and its secretion lacking; the use of thyroid extract is useful in such cases. Good fresh milk should be taken freely when the patient is able to to digest it. The disease is probably identical with the night-blindness accompanied by xerosis of the conjunctiva, which is so commonly seen amongst armies in the field and amongst others exposed to privations, and which is cured in its early stages by the use of liver in the diet. It is, however, a more advanced form of the condition, and, once it has been reached, the prognosis is much more serious."

At the risk of repetition, I might make a few further observations founded on cases seen locally. There is a certain amount of confusion between the condition described as epithelial xerosis and keratomalacia. No doubt in certain cases they tend to overlap, but there is not necessarily a triangular patch of xerosis nor yet white foam in keratomalacia, although such may occur. On the

other hand, the triangular or rounded islands of greasiness on either side of the cornea may occur in an apparently otherwise normal individual, without night-blindness, and the term epithelial xerosis seems to fit such cases. The smokiness of the conjunctiva appears to be about the earliest sign in the eye. Here it may be observed that darkening and thickening of the conjunctiva has been noted as a symptom of scurvy by Neil McVicar[8], in patients of all ages in South Africa. As distinct from the experimental condition the lids are not affected, except in so far as the palpebral conjunctiva is concerned, nor is there as a rule lacrymation or photophobia until the cornea is attacked. There is not necessarily any inflammatory reaction, and the vascularity of the conjunctiva is diminished rather than increased; quite a different picture from McCollum's experimental rats. There are certainly cases in which the cornea is infiltrated very early, with very limited conjunctival signs, but it seems to be rare for the cornea to be attacked first in humans, as it is in the experimental animals referred to by Professor Hopkins. The rapidity of the corneal destruction to which he refers is common in the clinical condition. The tears as a rule are scanty, and in very marasmic children are sometimes almost absent, but on the other hand they may be excessive in amount; in such cases there is a wet sodden condition of the epithelium with some conjunctival swelling and hyperaemia, less smokiness, and a wet running nose, quite a contrast to the dry greasy variety with marked smokiness. In the only case investigated the lacrymal secretion was found to be a turbid, alkaline, albuminous fluid, with 1.75 per cent. total solids, and 0.73 per cent. chlorides (as Na Cl)—a decided diminution in the chlorides as compared with the normal. Night-blindness is by no means constant, but it is frequent. There appears to be a tendency to overlapping between the fundus pictures seen in this condition, retinitis punctata albescens, and retinitis pigmentosa. In the first there is perhaps only a pale fundus with possibly a fine white mottling; in the second there may be a pale retina, pale disc, with faintly red vessels thereon (indicative of a watery blood stream), and a mid-peripheral belt of discreet white spots; in the third there may be but a pale yellowish disc, with fine arteries and a light or normal coloured retinal ground with fine white mottling and perhaps an isolated line of pigment along a vessel. In all there may be night-blindness. Here it may be mentioned that the bone-corpuscle type of fundus in retinitis pigmentosa is not common in Madras.

In bad cases associated with wasting, the nasal, buccal and laryngeal mucous membranes are affected. The intestinal mucous membrane seems to be one of the most important seats of trouble, for whether the child, with greasy, smoky, concentrically rippled conjunctiva, be covered by a loose bag of skin, or fat and well

nourished, there is generally a story of diarrhoea. In adults diarrhoea is the most common antecedent to a rapidly developing softening of the cornea. In children, broncho-pneumonia is sometimes an antecedent, and in severe cases frequently a terminal affection. A very important but inconstant feature is jaundice. Sometimes it is very marked and gives the scanty conjunctival débris in the fornix a greenish yellow colour. It appears to be more common in adults. It is sometimes associated with a small cirrhotic liver, at other times the liver is large. In one case a fairly well-to-do woman of middle age developed keratomalacia in association with an irregular, enlarged and painful liver, and intense jaundice, probably of a malignant nature.

Although the descriptions of experimental keratomalacia are somewhat meagre, there would appear to be considerable justification for the view that this clinical condition is not its counterpart. The latter is apparently much more complex, but may include such manifestations as are produced by deficiency feeding. In support of this view it may be stated that the rapid improvement produced in animals by feeding with substances rich in fat-soluble A has not been observed in human cases here.

With the idea of testing the value of introducing fat-soluble A together with protein into the diet, an experiment was undertaken in the second half of 1920. A fresh mixture was prepared daily consisting of yolk of egg 1 part, fruit juice 1, butter (ghee) 1, raw meat juice 1. Adults received 8 ozs., babies 2 ozs. per day. Eight patients were treated with this mixture, in addition to their food for over a month; all improved, but not very rapidly, or any more markedly than eight similar cases treated with cod liver oil and triple chloride mixture, as recommended by Colonel Kirkpatrick.

Seven of these experimental cases had a history of fever or diarrhoea occurring before the onset of eye symptoms, one had no history of previous trouble. Numerous others (fourteen in each group) started the treatment, but as it was not kept up for over a month they are not included. This experiment took up a considerable time. It was personally supervised by Captain Craggs, who has had a large experience of such cases during his association with this hospital for a number of years. At times the mixture had to be suspended for a few days on account of digestive trouble, but on the whole the feeding was regularly carried out. Without going into tedious details of the experiment, it may be stated that the result was negative. It may be that the quantity of fat-soluble accessory factor in this diet was not sufficient. Apparently this is a matter of great importance, as has been emphasised by Harriette Chick and Elsie Dalyell[2] in connection with their Vienna work.

It would be impossible to give a correct impression of keratomalacia as seen here, without drawing attention to the population

from which our cases are drawn, and the manner in which its individual members strive to exist on a minimum of food.

In Southern India the bulk of the poorer classes are always in, what McCollum terms, the " twilight zone." Not only is this so as regards fat-soluble A, but also as regards other vitamines, and the optimum ratios of food principles in general. The majority of our cases of keratomalacia are noted as taking " ordinary diet," which consists of rice wetted with pepper water, or a thin vegetable curry with few, if any, green leafy vegetables in it. Milk, meat, eggs and butter, as a rule take no part in the diet, or possibly meat and milk may be partaken of occasionally. In certain districts fish may be partaken of fairly liberally, but this does not seem to protect, contrary to the experience of Mori in Japan, who noted that fish eaters escaped. When one considers that such is the state of affairs amongst the poorer classes in Madras, it is not surprising that there are many apparent evidences of deficiency and insufficiency with respect to diet. One has but to observe the average cases attending a large out-patient department in this part of the world, to have this impression suggested to one's mind. Take, for instance, the Government Ophthalmic Hospital. Not only does one see 60 to 100 cases of keratomalacia per annum; one also sees early senility, stunted growth, early cataract, arterial degeneration, atrophy of the skin, low body weight, slow cerebration, unaccountable oedemas, beri-beri, and a large variety of fundus conditions presumably dependent on vascular degeneration. From a general point of view the chief feature in the picture is loss of elastic tissues and general atrophy. Atrophy of the skin, subcutaneous tissues and muscles is the simplest expression of this almost universal deficiency. More complex pictures are produced as the result of structural alterations in the blood-vessels, and still more complex when the simpler forms of degeneration such as fatty change, or colliquative necrosis settle upon a particular tissue, for example, the cornea. In the two latter groups there are possibly other factors at work besides mere deficiencies of food constituents, and one has to think of toxaemias and endocrine alterations, either primary or as the result of deficiency in the first instance. The fact cannot be lost sight of, that there are at least two other important (and exceedingly common), conditions to be taken into account when considering this picture of extensive degeneracy: syphilis and consanguinity.

Assuming the necessity for a food deficiency in keratomalacia, it is theoretically possible that this may occur at different levels. First, the intake may be deficient, all other conditions being normal. Secondly, the diet may be adequate, but the absorbing surface may be defective. Thirdly, the diet may be adequate, the alimentary canal may be healthy, but the organ or organs which

modify the products of digestion as absorbed from the blood stream may be at fault. As has been shown above there is ample opportunity for keratomalacia to occur at the first level amongst the poor in Southern India, but one would expect the disease to be more common if this were the only aetiological factor. As regards the second and third levels, it might appear at first sight that there are illustrative cases to be found. Numbers of our keratomalacia cases give a history of diarrhoea. What the exact nature of the disorder may be which is responsible it is hard to say, as no post-mortem evidence is available, but it is a very important factor in the condition as it is the most frequent cause of death. It is not an essential feature as cases occur without affection of the alimentary tract. The fact that in certain cases adults develop typical eye symptoms late on in an intractable intestinal disorder, having previous to the onset of the enteritis been in good health, is not necessarily explained by the deprivation resulting from the defect in the alimentary canal. The enteritis may have merely determined the onset of the keratomalacia in an individual previously on a border line dietary. The same applies to those occasional cases in which the onset of liver disease in an apparently healthy individual determines the onset of typical eye symptoms. The theoretical consideration of the possibility, however, draws attention to the great importance of the alimentary tract and liver. Enough has been said about the former to emphasise the rôle of intestinal disease, but the liver deserves a little further attention.

There are certain clinical observations which suggest that the liver plays a very important part in keratomalacia, but it cannot be assumed that this is more than secondary in its relation to the disease.

That cod liver oil gives equal results to butter fat in about the same amounts, leads one to enquire whether there is not something of importance in this oil derived from the liver itself. Mori draws attention to the value of liver feeding, and his observations are supported by the custom, which has long been in vogue on the Malabar coast, of feeding children suffering from keratomalacia on fried liver.

I have it on very good authority that the results of such feeding are very beneficial. It may well be that feeding with other glandular organs would act in the same way, but there is no evidence that this is so unless the administration of thyroid extract be taken as such. With reference to the association of liver and eye disease, Oatman[9] says, " hemeralopia, with and without pigmentation of the retina, has been observed in connection with chronic diseases of the liver." He also mentions that Landolt considered that an intimate relation existed between hepatic cirrhosis and diseases of the choroid and retina.

Although many of our cases of keratomalacia have liver disturbance, it is more than probable that in the majority of such cases the liver is secondarily affected. Cirrhosis of the liver is frequent in Madras. In a recent series of 836 post-mortem examinations at the General Hospital, it was present in over 5 per cent. Chronic intestinal disease is also common here, and cirrhosis appears to have a definite relation to catarrhal and ulcerative affections of the intestine. Cunningham[10] has called attention to the association of cirrhosis and jail dysentery in another part of India.

Since, as we have seen above, bad and insufficient food is almost universal amongst the poorer classes, we have locally all the factors necessary for the production of a vicious circle, such as one is accustomed to associate with keratomalacia, in which alimentary tract and liver play their part.

In conclusion the following summary may be given :—

There is in Southern India a clinical condition known by the name of keratomalacia, in which the symptoms of corneal degeneration form one of the leading features, inasmuch as it most frequently brings patients to seek relief; night-blindness is frequent but not so important a symptom.

The disease is not sharply marked off, but is a complex generalized disorder frequently associated with affections of the alimentary canal, grave constitutional disturbance, and marasmus.

In advanced cases it frequently ends fatally with terminal intestinal and lung affections. In less severe cases blindness is a common result.

It is practically always associated with deficiencies in the dietary of a general type, but general deficiency is so common, that to account for the comparative rarity of the disease one must suppose that other aetiological factors or determinants are necessary.

Clinical observations suggest the great importance of the liver, thyroid and intestine in these respects.

The condition does not appear to correspond to experimental keratomalacia or xerophthalmia, due to the deprivation of fat-soluble A, nor does it respond in the same way to the administration of a diet rich in this food essential.

It does, however, respond slowly to treatment, the chief measures being general care of the eyes and intestinal tract, and the administration of cod liver oil, liver, thyroid extract, and salts.

The prognosis as regards sight is bad if both corneae are attacked before treatment is commenced.

The condition is undoubtedly preventable, but the problem of its

prevention, like so many of the great health problems of Southern India, largely resolves itself into an economic one to be dealt with by the financial rather than the medical authorities.

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ANNOTATION

The Psychology of Vision

The science of psychology, for science it may now be called since its rescue by Darwin from the mediaeval burdens imposed on it by metaphysical philosophy, is the most important science of all, for all others, even philosophy, are subject to its laws. Like all other sciences its laws must be based on observed facts and not on mere plausible hypotheses. Ophthalmology is in a very special way able to be of use in this connection, and it was a happy idea to devote a discussion at the last meeting of the Ophthalmological Society of the United Kingdom to the subject of the Psychology of Vision in Health and Disease. The discussion was introduced by papers by Prof. C. Spearman and Sir John Parsons. Both of these papers are already so condensed by two such masters of the art of condensation as to make further condensation impossible; they must be read in full. The subsequent discussion maintained the high scientific standard set. Sir Robert Armstrong-Jones laid stress on the importance of feeling tone, what has been described as the hedonic tone in dealing with the psychology of vision. He pointed out that in every sensation there is an attitude of the subject in the presence of an object, each sensation having its own state of feeling tone. He gave some interesting details on the effects of colour on the mental patients at Claybury and the Maudsley Hospital.

Dr. Kinnier Wilson dealt at considerable length with the psychological peculiarities in certain visual auras in epilepsy of

the type described by Hughlings Jackson as a " voluminous " mental state. He divided this class of case into two main groups : (1) The familiarity type and its reverse or unreality type; (2) the visual memory type. These, he pointed out, must carefully be distinguished from the group of special sense auras described in a Bowman lecture by the late Sir William Gowers, although certain hallucinatory phenomena in the sensory sphere of taste and smell are associated with them, constituting what is known as the uncinate variety of epilepsy. He discussed the various psychological solutions suggested and said that the view he preferred was the one that takes cognisance of a separate " familiarity-quality " (*Bekanntheitsqualität*) as a psychical phenomenon attending perception. If Jackson's view that the state is not the result of epileptic discharge of the highest centres, but of over-activity of the next lower centres, as yet untouched by the discharge, were correct we should expect the phenomenon to be present in practically every case of epilepsy. While he did not wish to be taken as attempting to localise the physiological substratum of the " déjà vu " phenomenon in the cortex of the temporal lobes, he felt that he might go so far as to say that when, in idiopathic epilepsy, the discharges involve the neural elements of the temporo-sphenoidal lobes a peculiar mental state is particularly apt to develop; it may take, *inter alia*, either the " familiarity-unfamiliarity " form, or that of the " visual memory "; the latter results from excitation spreading to physiologically related centres, while the former has a special psychological quality of its own, not to be localised on the physiological level.

Mr. Percival continued the discussion, and although warned off mathematics by the secretary, was unable to resist the temptation just to mention the " innocent-looking " expression $\frac{d^3y}{dt^3} = \theta$. He pointed out that we might fairly consider the physico-psychical apparatus of vision as a perfect or reversible engine, and suggested that the absence of the nodes of Ranvier in the optic nerve might indicate that that structure was of a commissural nature. He next referred to the question of the oscillatory discharge of nervous current seen in tremors, and suggested that this phenomenon might afford an explanation of the curious fact that in optic nerve lesions the estimation of light difference is much more affected than that of the light minimum.

Mr. Juler's contribution to the discussion dealt with amblyopia from disuse. In order to get more exact data he had collected a series of cases of juvenile traumatic cataracts. The cases were 22 in number, and he considers that they give evidence in favour of amblyopia from disuse affecting the eyes of children up to six years of age, that it does not attack eyes after the age of seven years,

that it is not merely a standing still of the development of the retina or certain cerebral associations, but that it causes a real retrogression of the effective power of the retinal impulse on the consciousness, after the macula has reached an age at which central vision of 6/6 is normally present. A table with full details of the cases accompanies the report in the Society's Transactions.

Dr. Harford next discussed the relation of the psyche, soma and pneuma as forming the tripartite nature of man and illustrated his views by diagrams which are reproduced in the Transactions. He pointed out that while the psyche includes all that is expressed by the term "mind," it is also the domain of the emotions, the thoughts, the memory and the imagination, and is the recipient of the impressions which come to it from outside through the medium of the special senses and the director of the various activities of which the human body is capable. He followed McDougall in explaining the method of working of the psyche by classifying its activities under three heads, which may be described in their relation to vision.

1. Cognition, by which a visual image is recognized.
2. An affection, or the effect produced on the feelings or emotions as a result of (1).
3. A conation or the action of the will produced by (2).

He further illustrated his views as they applied to visual testing and coal miners' nystagmus.

Mr. Worth said he had never come across a case of false macula, that is, true fusion in an abnormal part of the retina, but that in a case of squint with no binocular vision and no trace of fusion sense, with good vision in the deviating eye, there was no suppression for moving objects in the part of the field of the deviating eye which was not overlapped by the fixing eye. As regards amblyopia ex anopsia he cited an involuntary experiment in which an eye, originally sound, was occluded in the treatment of squint and became nearly blind as a result of the occlusion being unduly prolonged, which had convinced him of its existence.

We hope that this very imperfect summary of a most interesting discussion may induce our readers to study the original with more interest than is usually accorded to the transactions of learned societies.

ABSTRACTS

I—IRRITATION OF THE CERVICAL SYMPATHETIC

Wölfflin, E. (Basle).—Clinical researches on irritation of the cervical sympathetic. (Klinische Untersuchungen über Halssympathicusreizung.) *Arch. f. Ophthal.*, Vol. C, 1919.

Wölfflin here reviews the facts already recorded, and adds some fresh observations about a condition to which little attention has hitherto been paid in ophthalmological literature as compared with paralysis of the cervical sympathetic.

The symptoms of irritation are (*a*) ocular, and (*b*) vascular. In the great majority of cases these are associated, while less frequently only those of the one or the other group may be present.

The ocular symptoms, in the order of their importance and frequency, are mydriasis, widening of the palpebral fissure and exophthalmos.

Mydriasis is always observed; it may be the only ocular symptom present. In the author's cases the pupil was not dilated *ad maximum* and reacted to light and on convergence, though more slowly than normally. He doubts the statement in Römer's text-book that absolute rigidity of the pupil may occur.

Widening of the palpebral fissure is nearly always present. This sign is best observed in the dark room by throwing the light with a mirror rapidly from one eye to the other, the difference between the two being then increased. When observed for some time in diffuse daylight, the variation in the distance of the upper lid margin above the limbus is often striking.

Exophthalmos is the least reliable sign, and when present is usually not marked. If it is not accurately measured its presence may be falsely inferred from mere inspection of the width of the palpebral fissure. While the other two signs each owe their origin to the action of one muscle, the processes concerned in the production of exophthalmos are more complicated, and the explanation of it is rendered more difficult by the fact that the results of experimental research have not been uniform, enophthalmos having been observed by some. Other symptoms of less regular occurrence are contraction of the conjunctival vessels, as well as those of the retina and uvea, most marked in the retinal arteries and veins; an increased flow of tears, a rise in the intraocular tension, and an increase in the dilatation of the pupil on pressure over the superior cervical ganglion.

The vascular symptoms are very variable. Typically they consist of pallor of the corresponding side of the face and lowering of the temperature, but the conditions may be the reverse of these,

accompanied by increased perspiration. In rare cases they are found on the opposite side of the face. In the experience of the author these symptoms are less constant and pass off more rapidly than is the case in paralysis of the cervical sympathetic.

The causes of irritation consist of a peripheral group (where compression of the cervical sympathetic is due to cervical tumours, enlarged lymphatic glands, etc., commencing apical tuberculosis, pleuritic bands, and trauma), and a central group, arising in a subcortical vasomotor centre in the mid-brain. In cases of central origin the vascular symptoms alone are usually present, and are elicited by various states of emotion, and in angina pectoris and allied conditions.

The diagnosis is usually easy, though sometimes more difficult than that of paralysis, if attention is directed to the oculo-pupillary symptoms alone. The prognosis is better than that of paralysis. In the majority of the central group of cases improvement or complete recovery ensues. In those of peripheral origin it depends on the early removal of the exciting cause.

As to treatment, apart from operative procedure, only galvanisation of the cervical sympathetic has hitherto been recommended.

THOS. SNOWBALL.

II—THE MICROSCOPIC STUDY OF THE LIVING EYE

Gallemaerts and Kleefeld.—The microscopic study of the living eye. (**Étude microscopique de l'oeil vivant.**) *Ann. d'Ocul.*, Vol. CLVI, November, 1919 (three photographic illustrations in text).

Gallemaerts and **Kleefeld** give an appreciative description of the corneal microscope of Czapski as used with the Gullstrand type of lighting, that is, the use of the Nernst lamp with a slit. The microscope is a binocular one, fitted with Porro correcting prisms and an adjustment for interpupillary distances of 56 to 76 mm.; various power eyepieces and objectives giving magnifications of 8 to 103 times, with fields of from 13 to 1.8 mm. in extent. The microscope is mounted on a stand resting on a table provided with a chin and head rest, with all the necessary adjustments. The luminous source is a horizontally-placed Nernst lamp fitted on a mobile stand, the lamp being placed in a tube whose far end is occupied by a vertically-placed adjustable slit with rounded corners. Between the light and the slit is a first condensing lens, on the far side of the slit is a movable disc pierced with three openings, a rectangular one identical with the longest diameter of the slit, the other two circular. By rotation of the disc each of these can

be brought successively into the axis of the tube. The light of the filament is condensed by a second aspherical lens of 7 cm. focal length and 50 mm. aperture, or of 6 cm. focal length and 43 mm. aperture, the more rounded surface being turned towards the lamp and the lens being furnished with a rectangular diaphragm with rounded corners similar to those of the slit. This second lens is placed in the line of the light emitted by the lamp, and can be moved to and fro on a 16-inch long arm projecting from beneath the lamp and moving with it. On this arm between the lamp and the lens can be placed coloured glasses or a parallel-sided cell with such fluids as fluorescein, eosin or methylene blue. The light should be directed on the eye to be examined at an acute angle with the direction of the microscope, and by the various special adjustments provided one can focus from the surface of cornea right down into the vitreous as required. The authors advise waiting for dark adaptation to occur before using the microscope in the dark room, as is necessary. The lighting is so regulated by the examiner that he obtains a slightly elliptical, uniformly lit, luminous band on the surface to be examined. When the higher powers are used more intense illumination is brought to bear on the eye by simple focussing. The size of the image is measured by an ocular micrometer, the depth by the vernier scale or graduated wheel attached to the substage.

The microscopic study of the living eye has been carried out very fully by Léonard Koeppe, who has endeavoured to bridge the gulf between ordinary naked-eye examinations and those of microscopic sections in this way and has recorded his observations in Vol. XCI et seq. of v. Graefe's *Archives*.

W. C. SOUTER.

III—GLAUCOMA AND EPIDEMIC DROPSY

Maynard, Lt.-Col. F. P., the late, I.M.S., (Crewe).—Glaucoma and epidemic dropsy. *Indian Med. Gazette*, August, 1921.

This is a report on 104 cases of glaucoma associated with epidemic dropsy in Calcutta. All but three of the cases were seen in an epidemic which occurred in 1909; and they included 22 cases recorded in the *Indian Medical Gazette* of October of that year. A few of the cases have been followed up for ten years or more.

Epidemic dropsy has been confused with the dropsical form of beri-beri. But **Colonel Maynard** agrees with Braddon, Manson and others, that it is a distinct disease, not due to vitamine-deficiency in diet. Anaemia is characteristic, mostly with oedema, particularly of the legs. Many of the cases begin with fever and

diarrhoea. There may be severe heart trouble, with risk of sudden death, though the mortality in some epidemics has been low. Maynard found the blood-pressure high. Several members of a household are usually attacked.

Of the patients affected with glaucoma 80 were male and 24 female. The average age was 34 years, only 19 patients being over 45 years old. This early age is noteworthy. The average time of onset of the glaucoma was about 5 months from the beginning of the disease.

The increase in tension was usually moderate, without shallowing of the anterior chamber or noticeable dilatation of the pupil. Congestion of the eye and pain were not very frequent, but the cornea was steamy in 49 cases, and coloured haloes were complained of in 73 cases. Contraction of the field of vision and pathological cupping of the disc were each present in about half the cases. The cupping developed rapidly.

Fifty-four eyes were operated upon, 34 by iridectomy, 18 by trephining, 2 by Lagrange's operation. Trephining was the most effectual.

H. HERBERT.

IV.—CONGENITAL WORD-BLINDNESS

Wallin, J. E., Ph.D.—Congenital word-blindness. *Lancet,* April 23, 1921.

Wallin is Director at the St. Louis Psycho-Educational Clinic and Special Schools. There is not a great deal that is original in his article as regards word-blindness itself, but he gives with particular clearness the position of word-blind children as pupils at school, and brings out their position as compared with the feeble-minded. While 85 per cent. of his cases were classified as subnormal in intelligence, only 5.2 per cent. were classed as feeble-minded. The article concludes with the practical point that word-blind children who are not feeble-minded should be assigned to special reading-disability classes, but the author admits that in St. Louis the financial difficulties have prevented any action being taken in the matter.

In the *Lancet* for April 30 there is a letter from Dr. Wood, of Rugby, referring to Wallin's article. This letter is rather misleading in one respect. The writer says, " I wish he (that is Wallin) had paid more attention to the question of inheritance, which was first dealt with by Mr. Sydney Stephenson, for to my mind this line of investigation will lead to a better understanding of the disease. A striking thing about Dr. Wallin's paper is the

close similarity between his figures of incidence and those of colour-blindness, and also the fact that the sex-incidence is the same. This similarity of incidence would suggest strongly that it is hereditary, and it would be informing to follow cases to see if the method of transmission in word-blindness follows the same course as that of colour-blindness." These remarks would lead one, the reviewer thinks, to suppose that Wallin had specifically referred to colour-blindness, but this is not the case. Wallin speaks of word-blindness being apparently to some extent sex limited. He says that word-blindness is about four times as prevalent among boys as among girls, but the suggestion as to any similarity between the two conditions is entirely Wood's.

Those interested in the educational aspect, more especially of the subject of word-blindness, would do well to read Wallin's article.

ERNEST THOMSON.

V—THE AETIOLOGY OF SPRING CATARRH

Gabriélidès, A. (Constantinople).—Etiology of spring catarrh. (Étiologie de la conjunctivite printanière.) *Bull. et Mémoires de la Société française d'Ophtal.*, 1920.

The author's researches on the above subject have already shown that eosinophile cells are present in excess in the conjunctival secretion, the tarsal vegetations and in the peripheral blood. In a further paper he has demonstrated that the disease is often present in more than one member of the same family, pointing either to a microbic origin or to a family idiosyncrasy. He has failed, however, to discover a specific organism in the secretions or vegetations and cultivations from the blood have also proved negative. Examined from the clinical standpoint the histories of 15 families are given, in each of which families two or more members are affected, the peculiar fact is brought out that it is usually two brothers or a brother and sister who have the disease, the other members of the family remaining unaffected; in other words, it does not seem to be contagious, and therefore is probably neither microbic nor parasitic, being rather of the nature of a personal malady affecting those members of a family who are specially susceptible. In order to test this theory he proceeded to inoculate a monkey by rubbing the tarsal proliferations into the scarified conjunctiva. The result after long observation was negative. He then inoculated the blind eye of a girl, but with the same result. He finally repeated the experiment on a small boy aged 6 from his brother aged 8 who was suffering from the disease. The result was again negative,

thus demonstrating that the disease is probably not due to an organism.

On the other hand, the eosinophile excess suggests that a toxic element is present. This excess occurs in many other diseases, including certain skin maladies, and the author thinks he can trace an analogy between spring catarrh and various skin affections, e.g., psoriasis, as in each case the action of the ultra-violet rays, and probably thermal rays also in the case of spring catarrh, is an important factor in provoking the onset of the disease. As a result of the irritation set up the cells secrete a toxin, against which the organism protects itself by multiplying eosinophiles. Viewed from this standpoint the affection of the tarsus is merely a symptom of a general malady which may sometimes be accompanied by enlarged glands, e.g., cervical or even axillary or inguinal, and occasionally there is such pronounced hypertrophy of the nasal mucous membrane that the disease has been described as " nasal conjunctivitis."

The author therefore arrives at the conclusion that spring catarrh is a manifestation of an auto-intoxication. The toxin probably exists in the serum and by stimulating the nerve endings provokes the pruritus which is a common symptom in this disease. As to the nature of the toxin and where it is produced he does not hazard an opinion.

CHARLES KILLICK.

VI—HISTO-PATHOLOGY OF TRAUMATIC CHOROIDO-RETINITIS PROLIFERANS

Mawas, M. J. (Paris).—Traumatic choroido-retinitis proliferans, studied histo-pathologically. (La chorio-rétinite proliferante traumatique. Etude histo-pathologique.) *Bulletins et Mémoires de la Soc. française d'Ophtal.*, 1919.

Mawas has been surprised, like other ophthalmic surgeons, by the frequency of choroido-retinal lesions accompanying wounds of the face or skull, with apparent integrity of the organ of vision. These lesions start by a more or less abundant haemorrhage which clears up in a few weeks' time. Its absorption is followed by the typical picture of traumatic choroido-retinitis proliferans usually found at the macula or surrounding the disc. The author has tried to solve the problem as to how these lesions have been brought about, to discover the relationship between the primary haemorrhage and the subsequent changes, the source of the effused blood, and to what extent they are curable.

They can be grouped according to the following table:—(1) Lesions of attack. This is the period of onset characterised by

haemorrhage from the choroid and rupture of the retina. (2) Lesions of defence or reaction which may last for several weeks; and (3) Lesions of consolidation, which is the last phase, characterized by absorption of the blood, cicatrization, and the fusing of retina with choroid.

Mawas states that the blood is derived from the lamina choriocapillaris. If the initial shock is not great the haemorrhage may be absorbed without any trace being left ophthalmoscopically, as it fails to penetrate the layer of pigment epithelium. Greater force, however, causes the epithelium to yield, though with difficulty, and the retina is detached in one or several places. Microscopic sections demonstrate that this primary rupture is situated at the macula where the retina is thinnest. Other retinal lesions, partly due to concussion and partly to dissociation produced by the red corpuscles, appear at the same time. These include necrosis of the layer of rods and cones and degeneration of the layer of ganglion cells, while the other retinal layers undergo a process of fusion. While these changes are taking place the haemorrhage is being replaced by a black pigment directly derived from it. The above phase constitutes the lesions of attack. The lesions of defence are due to an effort of the retina to protect itself by an intense phagocytosis of the haemorrhage and the necrosed elements, together with the proliferation of the pigment epithelium, the neuroglia and connective tissue, the result being the formation of the intensely white areas and patches of pigment, while in addition there is vascular congestion which produces an inflammatory oedema leading to a fibrinous exudate and diapedesis. Such are the main features observed microscopically.

The last phase or lesions of consolidation consist of a penetration of the vitreous by " young elements " (Mawas' own words*) and fibroblasts, which are gradually replaced by a dense connective tissue, which takes the place of the destroyed retina and forms a union between the vitreous proliferation on the one hand and the choroidal lesions on the other.

CHARLES KILLICK.

* He does not say whence derived.—C. K.

VII—SECONDARY GLAUCOMA

Abadie, Ch. (Paris).—Secondary glaucoma. (Des glaucomes secondaires.) *La Clin. Ophtal.*, February, 1921.

Abadie's article is somewhat discursive, but the reviewer wishes to bring forward an idea which seems to be original on Abadie's part. He says, " Contrary to what was formerly held, these glaucomas may come on without the least adhesion or the

least sub-iridal exudate. How are we to explain their appearance?" He proceeds to recapitulate the facts regarding the blood supply by reflex action, and points out that if the centripetal peripheral excitation becomes abnormal the centrifugal action caused by it becomes also abnormal and is followed by an abnormal circulation. "So when an infective process strikes the iris tissue it necessarily irritates in an abnormal way the circular sympathetic nervous plexus contained in it. This irritation of the sympathetic, transmitted to the ophthalmic ganglion or to the Gasserian ganglion, or possibly to other ganglia farther off, brings about reflex vaso-dilatation of the ciliary arteries, increased blood supply, increase of intraocular tension, and the glaucomatous state." Then again, after dealing with the effect of treatment, preventive and otherwise, Abadie asks, "Why does iridectomy cause the disappearance, in such a remarkable way, of these secondary complications (in this case it must be remembered that the glaucoma is the secondary complication referred to), the prognosis of which, without iridectomy, is absolutely hopeless." He answers his question in the following words, which the reviewer has thought should be carefully considered by ophthalmologists in order that their correctness may be confirmed or denied: "In removing a part of the iris tissue by iridectomy we are taking away a part of the sympathetic nervous tissue which it contains. We are, in fact, performing an iridal sympathectomy. We are cutting the nerve circuit. . . . Once this circuit is cut, things become normal, the peripheral centripetal irritation being no longer transmitted and the centrifugal vascular disturbance caused by it in turn disappearing."

ERNEST THOMSON.

CORRESPONDENCE

A PROTEST

To the Editor of THE BRITISH JOURNAL OF OPHTHALMOLOGY

SIR,—I feel sure that many of the shareholders in the company owning THE BRITISH JOURNAL OF OPHTHALMOLOGY will agree with me in entering a strong protest against the attack upon Dr. Edridge Green which appeared in the editorial columns of their journal last month. It is, moreover, inconceivable, at least to me, that this infraction of the dignity of a publication whose sole aim is the advancement of our science, could have appeared with your sanction, Sir, as Editor. Your accomplishments, as an experienced Editor, would, I am confident, have assigned the objection-

able annotation to its proper place, namely, the correspondence column, where the writer would have been required to acknowledge his identity, and yet the responsibility has been laid upon you of making a personal attack upon a colleague whose work has been received with the highest appreciation both in Europe and the United States. The result can only be that an entirely false impression will be created among our foreign colleagues as to the position which Dr. Edridge Green holds as an authority upon colour vision among his colleagues in this country. It will be a poor day for science if so reprehensible a means, as here portrayed, for expressing a difference of personal opinion, were to become common.

I am, Sir, Yours truly,

PERCY DUNN.

WIMPOLE STREET, LONDON, W.
March 8, 1922.

[We refer our readers to the annotation in question and leave it to them to judge if the above censure is justified. In our opinion the comments are a fair scientific criticism.—EDITOR.]

COLLOSOL ARGENTUM (FOR THE MEDICAL PROFESSION ONLY)

To the Editor of THE BRITISH JOURNAL OF OPHTHALMOLOGY

SIR,—In your last issue a letter from Mr. R. R. James calls attention to and criticises a circular booming the above drug in the cure of ophthalmia neonatorum by dropping a solution of it into the conjunctival sac. He asks if it has been tried at St. Margaret's Hospital for ophthalmia neonatorum. In reply, I may say that it has been extensively tried and found to be of little or no value in weak solutions, and in strong solutions causes pain and irritation without any advantage over other silver preparations. It is also our experience that unless silver preparations are rubbed into the conjunctiva they do not produce the desired result.

Yours faithfully,

M. S. MAYOU.

HARLEY STREET, LONDON, W.

To the Editor of THE BRITISH JOURNAL OF OPHTHALMOLOGY

SIR,—I am greatly obliged to Mr. R. R. James for calling my attention to some extracts from a paper of mine in the *British Medical Journal*, 1915, Vol. I, p. 104, published in a brochure

issued by Crookes Laboratories, which I had not noticed. I have written to the Crookes Laboratories and have received a reply as follows :—

LETTER TO CROOKES LABORATORIES.

43, PRYME STREET, HULL,
March 7, 1922.

Re COLLOSOL ARGENTUM.

GENTLEMEN,—My attention has been called to a brochure of yours on collosol argentum which I understand has been extensively circulated among medical men. You state that this is a plain statement of facts relating to the actions and uses of collosol argentum based entirely on the Published Reports of Medical Authorities in Standard Medical Journals. You quote from an article of mine in the *British Medical Journal*, 1915, Vol. I, p. 104.

Quoting me under gonorrhoeal ophthalmia, you say, " Patients treated with collosol argentum drops were cured in five days, and in severe cases which had failed to respond to silver nitrate and mercuric chloride, collosol argentum stopped the discharge in thirty-six hours and checked the ulceration, with the result that rapid recovery followed."

The natural inference from the above is that collosol argentum was substituted for the nitrate of silver and irrigations of mercury perchloride, whereas in the article it is definitely stated that the same treatment was continued, and, *in addition*, collosol argentum drops were introduced into the eye after each irrigation.

Again, quoting me in reference to purulent ophthalmia of infants, he states " that if collosol argentum drops were employed impaired vision or blindness from this cause would cease, and the drops being harmless to the conjunctiva the treatment of the case may be safely left to the friends."

Again, you make no reference to the fact that in the original article nitrate of silver drops and irrigations of mercury perchloride are used, and, in addition, collosol argentum drops are put in after each irrigation, and it is to the combined treatment that my remarks apply and not to the collosol argentum only, as stated by you.

Again, you make me say that the drops being harmless to the conjunctiva *the treatment* of the case may be safely left to the friends.

I said that " the drops being absolutely harmless may be safely trusted to the friends. The child is brought up once daily for the silver nitrate drops. The eyes are washed out every hour at home and the collosol drops put in." That is a totally different matter to saying that the treatment of the case may be safely left to the friends. As these quotations are so inaccurate and so grossly

misleading, in the public interest I must request you to stop the circulation of the brochure at once, and, in addition, to send to each person to whom you have sent the brochure, including myself, a full extract of that portion of my article referring to "Gonorrhoeal Ophthalmia and to Purulent Ophthalmia of Infants," with a suitable apology. I have only once trusted to collosol argentum entirely in the treatment of the above cases.

I enclose a reprint of my article as it appeared in the *British Medical Journal.*

I await your reply,

Yours faithfully,

A. LEGGE ROE.

REPLY FROM THE CROOKES LABORATORIES.

22, CHENIES STREET,

TOTTENHAM COURT ROAD, LONDON, W.C.1.

March 14, 1922.

A. LEGGE ROE, L.R.C.P., L.R.C.S.I.,

43, PRYME STREET, HULL.

DEAR SIR,—We learn with much concern and regret from your favour of the 7th instant, that a recently published brochure on collosol argentum has misquoted you in respect of your article of January 16, 1915. We would ask you primarily to acquit us of any intention to misrepresent or distort your views as expressed in the article above mentioned, and feel sure you will agree with us that there was not the slightest reason why such should have been made other than accidentally, as the addition of the omissions would hardly affect the value of your opinions.

Further, it would, we think, be taken for granted that practitioners who might be interested in cases similar to those mentioned in the brochure would, in the majority of instances, turn to the original article for explicit information, and for that reason care was taken that the authority for each statement should be quoted.

The Crookes Laboratories have issued to the medical profession an immense amount of literature from time to time, and this is the first occasion upon which, in spite of the many possibilities for error in the production of such material, that we have ever laid ourselves open to rebuke.

We hold ourselves to blame for not examining and comparing the manuscript of the brochure when returned to us by our printers, but you will appreciate that having regard to the care exercised in compiling from our available material it was assumed with certainty that all quotations were correct in every particular.

It would, unfortunately, be extremely difficult to trace all the medical men to whom a copy of the brochure in question has been sent already, but we can certainly assure you that no other copies will be issued containing the quotation to which you refer.

If we have your permission we should be glad of the opportunity of reprinting the whole of your article and issuing it with the brochure, calling attention to the discrepancies so that those interested may ascertain for themselves your exact statement.

We have been the more sorry for this occurrence as in the past your article has been of much value in securing the extended use of collosol argentum, which is now almost universally employed by ophthalmic surgeons. You will doubtless remember that you were practically the pioneer in utilising and publishing the results of your investigations, and we should, indeed, hold ourselves in contempt if any intentional action on our part was likely to cause inconvenience or trouble to a member of the medical profession to whose kindly interest we owe much.

Yours faithfully,

FOR BRITISH COLLOIDS, LIMITED,

(Signed) E. H. BUCKMAN, *General Manager*.

I am, Sir, Yours truly,

A. LEGGE ROE.

HULL, *March* 15, 1922.

SCLERO-CORNEAL TREPHINING AND HOLTH'S EXTRALIMBAL TANGENTIAL SCLERECTOMY

To the Editor of THE BRITISH JOURNAL OF OPHTHALMOLOGY

SIR,—Will you allow me some words in reply to Lieut.-Colonel R. H. Elliot's letter in your March issue (p. 141). He qualifies as a " serious error " my quotation of Duane's additional note in Fuchs's Text-book of Ophthalmology, 6th American edition, 1919, p. 512. I admit that I ought to have quoted literally " according to some " instead of " according to American operators," though Duane's additional notes generally concern experiences in America. I don't know if some of European statistics have " about 7 per cent " of late infections after sclerectomies, though Professor J. Meller, in his "Manual of Ophthalmic Operations," 1921, p. 333, speaks of the " terrific frequency " of this calamity after sclero-corneal trephining.

When Mr. Elliot writes: " Duane makes it absolutely clear that he is speaking of sclerectomy as a whole, and not of any single

operation, . . ." I must be permitted to present in italics Duane's first line in his note, quoted by Mr. Elliot himself in his letter: " (Sclerectomy, *more particularly in the form of the Elliot trephining operation,* . . .").

The fact that far more cases of late infection have been seen after sclero-corneal trephining than after any other form of sclerectomy is due not only to Elliot's operation having had the overwhelming majority of adherents but also to the fact that this operation—in spite of splitting of the cornea—gives a thinner conjunctival covering than the other sclerectomies. I saw myself not a single case of the late infection after my punch forceps sclerectomies from the anterior wound lip executed 1908 to 1913, but I have seen two cases later after the publication of Harrison Butler's " tragedy "; in these sclerectomies the scleral defect was very near the limbus.

I know no safer method of placing the anterior border of an effective scleral excision a good millimeter away from the limbus and by this means obtaining a thicker and better vascularized conjunctival covering than my tangential extra-limbal sclerectomy with the new 1 mm. punch forceps described in the December issue of this journal 1921, pp. 547-551. In Christiania since April, 1920, this operation is in constant use besides by myself by Professor Hj. Schiötz and Professor Sigurd Hagen in all the cases of primary chronic glaucoma which need operation. Till now no case has been followed by late infection; this may come later but I have very good reasons to believe much more rarely than after previous sclerectomies opening the anterior chamber.

Yours truly,

S. HOLTH.

CHRISTIANIA, NORWAY.
March 10, 1922.

NOTES

Deaths

As recorded in our last number Mr. Charles Wray has died in a London nursing home. We now give some details of his life and career. He was born in 1858 at Long Marston, Yorks, and was the only son of William Wray. He lost his mother when he was a few months old. In 1887 he married Henrietta Elizabeth, the second daughter of Henry Walmesley Welch, of Quebec. There were no children. He received his general education in Yorkshire and his medical education at the London Hospital. His expert knowledge of shorthand and drawing was of great assistance to him in his work. He was appointed ophthalmic surgeon to the Croydon General Hospital in 1887, and held the post until almost the last,

and there had a big practice. His private work was conducted in Croydon and in London. Mention should be made of the fact that he was attached to the Western Ophthalmic Hospital, and while there suggested a weekly evening opening of the clinic for the benefit of the working people who could not attend during the day, a suggestion justified by its success. His burial took place at Bandon Hill on February 16.

William Cochrane Murray, M.B., C.M., one of the surgeons to the Glasgow Eye Infirmary, died suddenly on March 12 at his Glasgow residence, 17, St. James' Terrace.

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Ophthalmological Society of the United Kingdom

AS already announced in these columns the Annual Congress of the Ophthalmological Society will be held on May 11, 12 and 13 next, at the Royal Society of Medicine. On Thursday, the 11th, the Edward Nettleship Prize will be presented, followed by the reading of scientific papers. At 2.15 p.m. there will be a discussion on industrial diseases of the eye (excluding accidents, miners' nystagmus and glass-blowers' cataract). The discussion will be opened by Dr. T. M. Legge, C.B.E., and Mr. Bernard Cridland. At 5 p.m. there will be a business meeting of the Society and members will dine together in the evening. The morning of Friday, the 12th, will be devoted to the reading of papers. At 2.30 p.m. there will be a clinical meeting at St. Bartholomew's Hospital. Members are invited to show cases of interest. At 8.30 that evening there will be demonstrations connected with tuberculosis of the eye. The morning of Saturday, May 13, will be devoted to the reading of papers.

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Appointments

Mr. P. G. DOYNE has been appointed assistant surgeon to the Royal London Ophthalmic Hospital. Dr. J. A. Pringle has been appointed honorary ophthalmic surgeon to the Kent and Canterbury Hospital.

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Oxford Ophthalmological Congress

THE next meeting of this Congress will take place on July 6, 7, and, if necessary, the 8th. On Thursday, the 6th, a discussion upon the significance of retinal haemorrhages will take place, to be opened by Dr. C. O. Hawthorne (London) and Mr. P. H. Adams (Oxford). The Doyne Memorial Lecture will be delivered on the morning of Friday, July 7, by Mr. J. Burdon Cooper, the subject being the aetiology of cataract. The official dinner of the Congress will take place on the evening of Friday, the 6th, in the Hall of Keble College.

Société française d'Ophtalmologie

THE next meeting of this Society will take place on Monday, May 8, at the Faculty of Medicine, Paris. The discussion, which will be opened by Dr. Dupuy-Dutemps (Paris), will be upon irido-choroiditis in the course of acute infections.

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The German Ophthalmological Society

THE German Ophthalmological Society meets in Jena on June 8, 9, 10. In addition to the usual business the bestowal of the Graefe Medal will be made. This medal is awarded once in ten years to the ophthalmologist, irrespective of country, who is regarded by the members of the Society as the greatest scientific benefactor to ophthalmologists. The previous recipients of the medal have been H. von Helmholtz, Theodor Leber, and E. Hering.

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British Journal of Ophthalmology, Annual General Meeting

THE Annual General Meeting of the shareholders of the Journal was held on Friday, March 10, at the Royal Society of Medicine (by kind permission of the Council of the Society). The audited accounts and balance sheet for the year ended December 31, 1921, were presented and adopted. The Report of the Directors, which was presented and accepted, disclosed the fact that the past year had been, financially, the most successful since the establishment of the Journal, and the Directors were able, for the first time, to declare a dividend. The Directors expressed their indebtedness to the Editorial Committee, the Editor and Sub-Editor, the Manager, and the Printers for their labours, and for the care and attention devoted to the interests of the Journal during the twelve months under review. A vote of thanks to the Board of Directors terminated the meeting.

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North of England Ophthalmological Society

AT the meeting of the Society held on March 20, 1922, the following gentlemen were elected to the Committee of the North of England Ophthalmological Society :—President, Mr. G. H. Pooley, Sheffield; vice-president, Mr. Thomson Henderson, Nottingham; treasurer, Mr. Harry Lee, Leeds; secretary, Mr. Percival J. Hay, Sheffield; members of Council, Mr. J. Gray Clegg, Manchester, and Mr. A. L. Whitehead, Leeds.